tissue change by continuity of structure, yet that extension is limited and that subsequently the normal tissue elements tend to be restored. The removal of degenerated myelin and the absorption of young connective tissue is he considers no more wonderful than the absorption of provisional callus round a fractured bone. He further says, we know that lead, mercury, etc., may produce results on the peripheral nerves exceedingly like those produced by alcohol and just as with alcohol if the dose of poison be too severe, death may ensue; but it is equally true that if the irritant noxa be withdrawn and food assimilated there is a tendency to complete restoration. In the recovery of alcoholic neuritis he considers the great factor to be, (1) the withdrawal of alcohol, (2) the administration of food, (3) time and the natural tendency to elimination and repair. With respect to other results believed to be brought about by chronic alcoholism, he says, there is no doubt that its existence makes the outlook much more serious in many acute and chronic diseases and it often makes a great difference as to chances of recovery. But we have attributed too much to its influence in the etiology of other diseases, especially to kidney disease and disease of the large and middle arteries. It is, he considers a most significant fact that in a large number of cases of alcoholic cirrhosis of the liver there is no albumen in the urine. But if alongside of chronic alcoholism there are other existing causes of degeneration, as valvular disease, or atheroma or granular kidney, then, he says, it is futile to expect that in such cases merely taking away stimulants will cure. A. F.

Beri-Beri.—(W. Duncan Scott, M.D., Edinburgh. The Practitioner, May, 1892.) We may attribute this disease, consistently with all available evidence, to a blood change, with symptoms due to the presence of an alkaloidal substance and an excessive acidity of the blood. The mode of production of this substance has not been ascertained. While it is possible that beri-beri is caused by a change, *per se*, in the constitution of the blood, resulting in the formation of a specific poison, it is equally, if not more probable, that this poison is the product of a micro-organism which finds in the blood, after exposure to certain climatic and meteorological conditions, a suitable *nidus*. All the conditions under which beri-beri occurs are favorable to the supposition that an ascomycetes (Lacerda) which may be in the soil, or the food (rice or fish) is the real cause of the disease. But others attribute it to a diplococcus (Peckelharing) a bacillus (Ogata), a spirillum (Taylor). This micro-organism may, so long as it exists only in the blood, cause wet beri-beri. It generates a poison which acts like muscarine and originates an excessive acidity along with disordered action of the sweat-glands and kidneys. At the commencement of the disease this poison may be eliminated by oxidation by means of iron or by improved general wet abolism. In later stages it is best neutralized by potash and ammonia, and it may be excreted by stimulation of the urinary tubules, while its specific action on the blood vessels is counteracted by atropine, and œdema prevented. The accumulation of this poison may also be checked by restoring the sweat-glands to activity. After a more prolonged infection a chronic neuritis is set up, accompanied by paræsthesia and anæsthesia and by paresis of the various peripheral nerves of the phrenics, vagus, etc. In these later stages this neurites is very similar to that caused by poisoning with carbon bisulphide, alcohol, etc., and the noxious substance no longer causes any symptoms of blood degeneration. The treatment then required is only that of neuritis and debility. Microscopically the author saw in the popliteal nerve of a case of dry beri-beri an interstitial infiltration consisting of a few scattered foci of small cells, showing abundant nuclei, but not of sufficient extent as to be pronounced interstitial neuritis. The dorsal spinal cord of another case showed a similar appearance. There was also congestion of the vessels of the kidney and the secreting epithelium showed cloudy swelling and some fatty degeneration. In neither case was it possible to see a micro-organism or microphyte. A.F.

Hydatid of the Brain.—Ll. D. Parey. (Australasian Medical Gazette, Aug., 1892.) A boy nine years old began to complain of dimness of vision, giddiness and headache. Gradually the headache became more severe, were paroxysmal and referred to the frontal and apical regions, and he frequently vomited food. Later, hæmiplegia and total blindness appeared about the same time. The patellar reflexes were diminished. No ankle clonus. The pupils widely dilated and insensible to light. Both optic discs atrophied, with veins enlarged and arteries contracted. There was a peculiar hollow note on pecussing the scalp over the upper part of the Rolandic area, one and half inches to the right of the median line. Operation was performed and the cyst re-